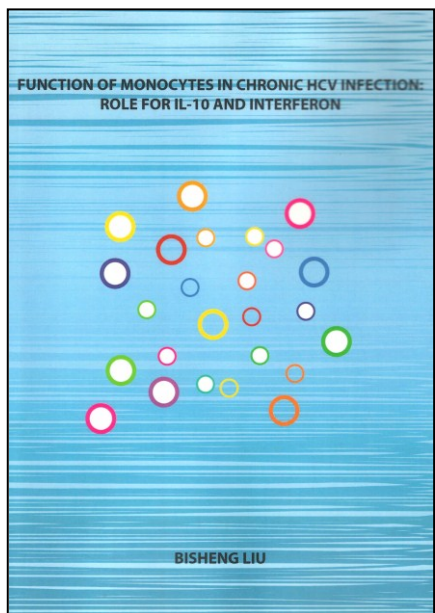




Nederlandse  
Vereniging voor  
Hepatology



## Samenvatting proefschrift Bisheng Liu

**'Function of Monocytes in Chronic HCV Infection: Role for IL-10 and Interferon'**

**Promotiedatum: 16 november 2011  
Erasmus Universiteit Rotterdam**

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Hepatitis C virus (HCV) is serious and growing threat to public health, affecting approximately 200 million people worldwide. The infected people are at high risks for liver cirrhosis and hepatocellular carcinoma – thus becoming a leading cause for liver transplantation.

Therefore, a good understanding of human immune system upon chronic HCV would be of great importance to fight against the virus. Currently, a clear understanding of the innate immune system in chronic HCV patients is not completely obtained. Our research have found that monocytes from chronic HCV infected patients have impaired ability to respond to bacterial-derived pathogens, which could be explained by the consequence of high level interleukin10 (IL-10), a potent immunosuppressive cytokine, produced by monocytes (J Leukoc Biol, 2009, 2011; Mol Immunol, 2011).

Type I interferons (IFN) form the backbone of current therapy for chronic HCV patients. However, only about 30-50% of chronic HCV patients respond to IFN-based therapy. Our research have suggested a likely explanation for this: one of the effects of human APC with type I and III IFN is to promote the cells' sensitivity to IL-10, a broad immunosuppressive cytokine. These findings are highly relevant to further improve IFN-based therapy for chronic HCV patients. The findings of this research have been submitted to renowned international journal for publication.

Numerous publications have reported that the IL28B SNPs predict IFN-based therapy for chronic HCV patients, and to understand the underlying mechanisms and the function of IFN $\lambda$  are of great interest of current research in the field of HCV infection. In the project, we have found for the first time that only human macrophages, but not monocyte nor dendritic cells, respond to IFN $\lambda$ . Furthermore, we found that IFN $\lambda$ , different from IFN $\alpha$ , enhance IL-12 production by pathogen-challenged macrophages. The importance of this research is acknowledged by our publication in BLOOD. ◀

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Aan de publicatie van dit proefschrift werd een financiële bijdrage geleverd door de  
Nederlandse Vereniging voor Hepatologie.

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